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Advances in wheat breeding for resistance to Fusarium head blight

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Abstract: Fusarium head blight (FHB), mainly caused by *Fusarium graminearum*, is one of the most devastating diseases of wheat globally. FHB causes an extensive reduction in yield and reduces the grain quality through its contamination with Fusarium toxins such as deoxynivalenol (DON), T2 toxin, HT-2 toxin, nivalenol, and zearalenone. This review provides an overview of updated progress of genetic studies on the resistance to FHB, with an emphasis on the sources of resistance to FHB, resistance gene/quantitative trait loci (QTL) mining, resistance gene cloning, major FHB resistance genes/QTL identification by molecular markers, and resistance mechanisms. The achievements of resistance breeding based on phenotype selection and molecular markers was also summarised. Based on the systematic analysis of breeding limitations and utilisation of FHB resistant materials, the authors put forward three suggestions: First, to toughen the resistance identification of wheat, testing traits such as Fusarium damaged kernel and DON need special attention as visual symptoms are less reliable, resistant varieties should be popularised, and the screening the resistant genes should be strengthened; The second is to use the additive effect of quantitative resistance genes accumulated from existing varieties to reduce the cost of resistance in order to create high yielding resistant varieties. Thirdly, to enhance research and utilization of new genes.

Keywords: breeding strategy; QTL mapping; resistance gene; resistance mechanisms; wheat scab

Fusarium head blight (FHB) is one of the most chronic fungal diseases in most wheat-growing regions globally. FHB causes an extensive reduction in the yield and deteriorates the seed quality through its contamination with a plethora of mycotoxins, in turn limiting its use for animal and human consumption (Schmale & Bergstrom 2010). The predominant pathogen of FHB is *Fusarium graminearum* (anamorph) Schwabe [teleomorph: *Gibberella zeae* Schw]. Other pathogens show similar symptoms less frequently with FHB include *F. asiaticum*, *F. equiseti*, *F. pseudograminearum*, *F. avenaceum*, *F. cerealis*, *F. sporotrichioides*, *F. avenaceum*, *F. poae*, *F. Sambucinum* and *F. culmorum* (Ji et al. 2019; Yun et al. 2019; Fabre et al. 2020). The first symptoms of FHB on wheat spikes occur shortly after flowering; as diseased spikes display premature bleaching and, if the weather conditions are suitable to FHB, pink-red mycelium and fungal conidia develop on the spike and the infection spreads by wind and rain-splashes

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to the entire spike of wheat as the pathogen grows and finally the infected kernels become shrivelled and chalky white in colour (von der Ohe 2010). Warm and moist weather is very conducive to the germination and invasion of the ascospore and a successful invasion results in killing the host plant (Geng et al. 2014). *F. graminearum* produces both ascospores and macroconidia: sexual and asexual spores, respectively (Schmale & Bergstrom 2010). FHB causes tremendous economic losses and reduces the quality and quantity of the wheat grain or even the failure of the seed formation (Ma et al. 2020; Khan et al. 2020). Yield losses due to FHB rank second after leaf rust and are particularly high in Canada, China, the US Midwest, Paraguay, southern Brazil, Uruguay, and Argentina. (Buerstmayr et al. 2019). For example, FHB caused 30–70% yield losses in Argentina in 2012 (Palazzini et al. 2015; Laraba et al. 2017), 11.6–39.8% of yield losses between 2000–2010 in Brazil (Reis & Carmona 2013), and 10–70% of yield losses have been reported in China (Zhang et al. 2011; Yun et al. 2019). Wheat grains infected by *Fusarium* spp. produce a plethora of mycotoxins, mainly deoxynivalenol (DON), the T2 toxin, the HT-2 toxin, nivalenol, and zearalenone (Miller & Ewen 1997). These toxins are among the fungal mycotoxins that cause deleterious health effects to humans and livestock, and make the grain unfit for international markets (Foroud et al. 2019; Khan et al. 2020). DON mainly produced by *F. graminearum* is the most potent inhibitor of eukaryotic protein biosynthesis by binding to the ribosome and can transport and affect the dopaminergic receptor in the brain (Herter et al. 2019). Fungicide classes including Tebuconazole, Triazoles, and Prochloraz are known as the most effective chemicals to prevent and suppress the FHB pathogen and reduce Fusarium toxins that contaminate wheat grains despite the resistance and environmental concerns.

Hence, the use of genetic resistant cultivars is an appropriate strategy. Traditional cross breeding involves the repeated evaluation of breeding lines under field or greenhouse conditions. This method is normally labour-intensive, time-consuming, and liable to be influenced by environmental conditions. Thus, it is appropriate to supplement the phenotypic selection with marker-assisted selection for FHB resistance (Anderson et al. 2007). In the last three decades, the search for host plant resistance based on phenotypic selection has made excellent progress in producing several wheat breeding lines with the best levels of FHB resistance. However, this has been hindered by the complexity of resistance genetics and the deficiency of the available FHB resistant germplasms. Besides, recent reports showed that among the five resistance types, type I and type II resistances are controlled by distinct genetic factors (Ma et al. 2020). FHB resistance in wheat is controlled by polygenics and is prone to be influenced by the environment (Bai & Shaner 1994). Besides, the expression of resistance is highly dependent on the environment, the pathogen, and the host (Randhawa et al. 2013), this is another challenge added to the complexity of breeding and phenotypic selection. To obtain improved cultivars resistant to FHB, many resistant identification methods have been used and a large number of breeding resources have been screened using molecular markers in the last decades. More than 400 resistant quantitative trait loci (QTL) have been identified so far (Buerstmayr et al. 2009; Jia et al. 2018; Ma et al. 2020) mainly located on chromosomes 5A, 3B, 6B, 6D, and 7D (Lin et al. 2004; Ren et al. 2019). The first QTL for type II resistance was identified in the Chinese wheat Sumai 3 on chromosome 3BS. This QTL was characterised by molecular markers and named *Fhb1* (Guo et al. 2003). *Fhb1* has recently been cloned from Sumai 3 (Rawat et al. 2016). QTLs, named *Fhb*2 and *Fhb4* were found to confer type II resistance have been identified and located on chromosome 3BS and 4B, respectively (Xue et al. 2010). The identification of FHB-resistant genes by molecular markers, the mapping of FHB resistant QTL (Cuthbert et al. 2007; Qi et al. 2008; Guo et al. 2015; Li et al. 2019a; Steiner et al. 2019), and the cloning of *Fhb1* and *Fhb7* (Li et al. 2019a; Wang et al. 2020), opened opportunities of getting easy-to-use markers and facilitated marker-assisted FHB resistance breeding. As a result, significant noble FHB resistant sources have been obtained thus far.

Progression wheat breeding for resistance to FHB

Sources of resistance breeding to FHB. The resistance sources to FHB with the most potential are innate (native) sources. They are more suitable to use compared to non-native sources due to their better agronomic and higher adaptability and post-harvest quality traits. In the early 1920s, screening of germplasms for FHB resistance was conducted for the first time in the United States (Atanasoff 1920) and resistant germplasm for breeding purposes have been distributed since 1985 (large trial network

https://doi.org/10.17221/1/2022-CJGPB

of the US wheat and barley scab initiative) (https://scabusa.org/). The most widely known native FHB resistance cultivars include Coker 9474, Massey, Ernie, Goldfield, Foster, Hondo, Patton, Roane, Heyne, McCormick, Wesley, NC-Neuse, Truman, INW0304, IL94-1653, Bess, Cecil, NY88046-8138, INW0411, and USG3555 (Liu et al. 2007; Murphy et al. 2019; Zhu et al. 2019a).

In China, the collection, identification, and application of wheat germplasm resources resistant to FHB have been developed since the 1940s (Ma et al. 2019). Improved and local varieties are the two main types of wheat FHB resistance sources that are widely in use nowadays. Sumai 3, Jingzhou 1, Wangshuibai, Wuhan 1, Baisanyuehuang, *Haiyan* species, and their derivatives, etc., are examples of the improved local varieties (Zhang et al. 2018). The local varieties are the wheat-related species resistance germplasms, such as *Leymus*, *Elytrigia*, and *Roegneria*. The first and largest germplasm screening for resistance of wheat to FHB was conducted in 1974 on 34 571 accessions composed of 23 434 domestic, 9 184 common wheat, 1 557 other rare resources of *Triticum*, 26 *Aegilops*, and 170 triticale materials. Of the 1 765 germplasm resources with intermediate resistance or above, 92.2% were Chinese, 71.2% were improved varieties and 28.8% were local varieties (Cainong et al. 2015; Guo et al. 2015: Ma et al. 2020). From the common wheat and landraces, 5.2% of the accessions were identified as moderately resistant.

This was the most comprehensive identification and screening of wheat FHB resistance sources in the world. It set a good foundation for wheat resistance breeding in China and the world (Li et al. 2019a). 2 141 wheat lines from CIMMYT were evaluated for FHB resistance in Shanghai and no resistant line was found, the percentage of moderately susceptible lines was less than 1% (Wang et al. 2018). Wan et al. (1987) studied the level of resistance to FHB in 1 076 wheat cultivars from China and abroad and only 30 wheat cultivars showed resistance. Ninety-five landraces, (64 Chinese accessions, 24 Japanese accessions, one Korean cultivar, and six cultivars from the USA) were evaluated for three types of FHB resistance in repeated experiments and most of the accessions (63.2%) were moderately resistant (MR) or moderately susceptible (MS) (Yu et al. 2008). The resistance of 762 wheat varieties in the Huang-Huai wheat region to FHB was identified by surface inoculation. 148 varieties with moderate resistance to FHB were selected, accounting for 19.4% of the tested varieties. Furthermore, only ten stable wheat varieties with moderate resistance to FHB were selected by single flower drip inoculation (Zhang et al. 2020).

Wheat landraces and wild relatives can also be a good source of novel FHB resistance. The most potent levels of FHB resistance were identified in *Leymus racemosus* (Tein.) Tzvelev (syn *Elymus giganteus* Vahl.), *Roegneria kamoji* (Ohwi) Ohwi ex Keng (syn. *E. tsukushiensis* Honda), and *R. ciliaris* (Trin) Nevski (syn. *E. ciliaris* (Trin) Tzvelev) (Weng & Liu 1989; Chen et al. 1995, 2005; Weng et al. 1995). A study on FHB resistance and the cytogenetics of intergeneric hybrids of *Triticum aestivum* L. with *Roegneria c.* Koch (*Agropyron*) species revealed that *Roegneria ciliaris* (*Agropyron ciliare,*) and *R. kamoji* (*Agropyron tsukushiense* var. *transiens*) were highly resistant to FHB (Weng & Liu 1989). *Fhb7* was found in *Thinopyrum ponticum* (Guo et al. 2015). Seven moderate resistance and five high resistance varieties were identified from the *Aegilops tauschii* accessions (Brisco et al. 2017). *T. carthlicum*, *T. dicoccoides*, *T. dicoccum*, *T. durum*, *T. polonicum*, *T. turanicum*, *T. paleocolchicum*, and *T. turgidum* are the core collections of tetraploid wheat subspecies, and various unique genes resistant to different major wheat diseases and insect pests have been transferred from these subspecies into common wheat and are widely used in wheat breeding worldwide (Fedak 2015).

For example, many accessions of *T. dicoccoides* (Oliver et al. 2007), *T. dicoccum, T. carthlicum* (Oliver et al. 2008), and *T. polonicum* were identified to have moderate to high levels of FHB resistance. Other *T. dicoccoides* accessions that carry moderately resistant genes include Mt. Hermon#22 and Mt. Gerizim#36 (Buerstmayr et al. 2003), PI478742 and PI481521 (Kumar et al. 2007), the tetraploid lines Tunisian 7 and Tunisian 34 (Huhn et al. 2012), and some *T. carthlicum* accessions for Type II resistance, Td161 for the overall FHB resistance (Buerstmayr et al. 2012), and *T. dicoccum* BGRC3487' for type I resistance. The homologues of *Fhb7* were detected in several genera in *Triticeae*, including 37 *Thinopyrum*, *Elymus*, *Leymus*, *Pseudoroegneria*, and *Roegneria* (Guo et al. 2021). Seven FHB resistance genes have been identified and have been designated as *Fhb1-Fhb7* so far, of which, *Fhb1* and *Fhb2* are derived from Sumai 3 (Cuthbert et al. 2006, 2007), *Fhb3* is derived from *Leymus racemosus* (Qi et al. 2008) *Fhb4* and *Fhb5* are derived from Wangshuibai and Sumai 3 (Xue et al. 2010), *Fhb6* is derived from *Elymus tsukushiensis* (Cainong et al. 2011), and *Fhb7*

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is derived from *Thinopyrum ponticum* (Guo et al. 2015). The selection and creation of new wheat materials with resistance to FHB by a relative species can provide new germplasm resources for wheat breeding.

Methods of investigating physiological resistance to FHB. The resistance types can be evaluated by using different inoculation methods. Type I resistance can be evaluated by the method described by Yoshida et al. (2007). In short, the inoculation can be performed at the flowering stage and the pathogen suspension should be applied at the centre of each spikelet, and the temperature (18–25 °C) and relative humidity (90–100%) for the inoculated spikes should be adjusted to facilitate the invasion of the pathogen for at least 24 h. The percentage of the diseased spikes can be determined visually based on a 0–9 scale following the method described by Patton-Ozkurt et al. (2009). *Fusarium* sp. infection to wheat can be controlled using morphological and physiological mechanisms (Gilsinger et al. 2005). Plant morphological traits involve the adjustment of the time and conditions that are not suitable to cause infection. The heading date, flowering time, plant height, (Buerstmayr & Buerstmayr 2016), and the presence of awns (Mesterházy 1995) have been associated with type I resistance and FHB severity (Gilsinger et al. 2005). The physiological approach, on the other hand, involves the utilisation of biochemical pathways to secrete chemicals that have the potential to suppress the growth of the pathogen after the initial infection (Mesterházy et al. 1999). Some other more stable FHB resistance QTLs occur at the same location with major genes such as *Rht8* (McCartney et al. 2016), *Vm-A1* and *Rht-D1*(He et al. 2019). The *Rht1* and the *Q* genes affects the flowering, plant height and spike related traits. Generally, type I resistance is common in barley and rare in wheat. It is mainly contributed by the spike morphology (Mesterházy 1995), and activated by the innate immune response (Foroud et al. 2012). More studies are needed on the role of type I resistance to FHB.

Type II resistance is usually evaluated by a single floret (spikelet) inoculation that injects an inoculum onto a central spikelet of a spike (point inoculation) both in field and greenhouse experiments and expressed as the spread of the infection within the spike. Several previous studies reported that type II resistance can cause the cell wall thickening of wheat rachis nodes and result in mycotoxin decomposition (Li et al. 2017), the most stable and utilised type of resistance attributed by various resistant genes. Generally, spraying the inoculation is considered as a way of measuring the overall or total resistance of wheat to FHB (Mesterházy 2017), effective for type I and type II resistance, but it is also appropriate to provide grains for evaluating the FHB resistance, DON resistance to kernel infection, and also the tolerance, as explained by Mesterházy (1995) and Mesterházy et al. (2015). Type III resistance is highly correlated with type I and type II resistance and it is usually used to describe the resistance to the mycotoxin accumulation (Rudd et al. 2001).

In addition, the used threshing method significantly affects the accuracy of the type III resistance assessment. During harvesting time, Fusarium-damaged kernels (FDK) that have a high level of DON can be blown out (Mesterházy et al. 1999), hence, the DON measurement may not represent the actual amount of DON produced in the cultivars being tested. Besides, a low FHB tolerance and FDK were described as two additional types of resistance (Mesterházy 1995). In a four-year experiment, the correlation among the FDK, FHB, and DON was determined all being significant at $P = 0.001$. The FDK/DON correlation was much closer than the FHB/DON correlation (Mesterházy et al. 2018). The issue of food safety cannot be identified from the FHB disease index and FDK result. Without identifying the toxin level, the safety factor cannot be detected and identified from the cultivar's data (Mesterházy et al. 2015). Cuthbert et al. (2007) defined the FKD as the kernels that are shrivelled, light weight, and chalky white or occasionally pink. The frequency of FDK can be calculated as the number of damaged kernels among the total examined kernels per plot. A negative correlation between the FHB disease resistance and the DON grain contamination was reported by previous studies (Bai et al. 2001), but not in others (Mesterházy et al. 1999; Wiśniewska et al. 2014). Another study conducted by Somers et al. (2003) suggested that the DON accumulation and FHB resistance might be controlled by independent genetic factors, whereas other studies reported that type II resistance might be more associated with a low DON content (Lemmens et al. 2005; Chen et al. 2006). The discrepancy among studies is that most of the FHB resistance evaluations focused on the visual head symptom, and cultivars that have low visual symptoms may be discarded due to the high percentage of FDK. In most cases, FDK and DON are generally neglected. This could be one reason that the visual FHB index and the FDK do not cor-

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relate. The tissue and assay method used for the DON evaluation, the threshing method, the stage of plant development during FHB infection, the degree of FHB resistance of evaluated genotypes, the disease development and factors related to the inoculation time/method could be another reason (Mesterházy et al. 2015).

DON and FDK were introduced as additional resistance types by resistance to DON and resistance to kernel infection (Bai et al. 2001). Therefore, analysing the response of cultivars to different parameters is needed as it may affect one's breeding method. The data on DON is very important when it comes to food safety. Some lines showed a low field FHB index due to type I and type II resistance that resulted in fewer kernel infections and lower DON content levels (Bai et al. 2001). Type V resistance is evaluated by measuring the DON content in the harvested grains using an enzyme-linked immunosorbent assay (ELISA) (Qiu et al. 2015). Previously, Ran et al. (2013) reported that determining DON using ELISA is the most preferred due to its specificity, stability, simplicity, and high throughput, but is only used for one mycotoxin, which means it can be used for a limited number of antibody-binding sites. In addition, ELISA is less accurate and sensitive than conventional chromatographic assays. Most advanced and powerful chemical analysis methods, like gas chromatography-mass spectrometry (GC-MS) or liquid chromatography-mass spectrometry (LC-MS), are used by many labs due to their higher accuracy. Type V resistance is important to the grain end-use quality, such as protein and starch qualities (Rudd et al. 2001). In general, the purpose of quantifying the DON content in the harvested product that would be processed into feed or food is very crucial. The DON content usually shows a significant positive correlation with the fungal biomass (Snijders & Van Eeuwijk 1991).

Mapping and cloning of FHB resistance genes

Mapping of FHB resistance QTLs. The resistance genes of FHB can be mapped by a monosomic analysis, chromosome substitution, and QTL mapping. Studies in literature and review articles on germplasm evaluations, marker assisted selection (MAS), and QTL mapping have been thoroughly discussed (Buerstmayr et al. 2019). *Fhb1*, *Fhb2*, and *Fhb3* conferring type-2 resistance were mapped to the wheat chromosome arms of 3B and 6B (Cuthbert et al. 2007). *Fhb4* and *Fhb5* conferring type-I resistance were mapped to the chromosome arms of 4B and 5A, respectively (Xue et al. 2010). Yu and Wang (1991) used monosomic analysis methods to locate the Pinghujianzimai, Honghudataibao, Yangangfangzhu, and Wannian2 FHB resistant genes. The FHB resistant gene in Pinghujianzimai was located on chromosomes 6D, 7A, 3B, 5B, 6B, 5D, 1D, 2B, and 3D, and the FHB resistant gene in Honghudataibao was located on chromosomes 5D, 7B, 1B, and 4D, conferring moderate resistance, whereas, the FHB resistant gene in Wannian 2 were located on chromosomes 4D and 5A. Likewise, the resistant genes against wheat FHB in U-136.1 were located on chromosomes 6B, 5A, 6D, 1B, and 4B, and the susceptible gene was located on chromosome 2B. The FHB disease resistance genes in Wangshuibai were located on chromosomes 5A, 7A, 4B, 6B, and 5B. Cai et al. (2019) have identified 31 QTLs from five recombinant inbred line (RIL) populations, 19 of them were mapped independently on chromosomes 1A, 2B, 2D, 3A, 3B, 3D, 4A, 4B, 4D, 5A, 5D, 6A, 6B, 6D, 7A, and 7D. The QTL on the chromosome arm 3AS and at the distal end of chromosome 3BS were mapped in Baishanyuehuang × Wheaton, Huangcandou × Jagger, and Huangfangzhu × Wheaton carrying both QTLs and Wangshuibai × Wheaton and Haiyanzhong ×Wheaton carrying one of the QTLs (Cai et al. 2019). The wheat FHB resistant genes of Frontana were located on chromosomes 3A, 6A, and 4D, and the susceptible genes were located on chromosomes 2A, 2B, 4B, and 7B by analysing the resistance of the population from the cross with the susceptible monomer cultivar Chris (Berzonsky et al. 2007). Yan et al. (2021) investigated the genetic bases of Y158 for FHB resistance and six QTLs with better resistance were identified. Among these QTLs, one was for type I resistance (Qfhi.nau-2D) and one was for type II resistance (Qfhs.nau-2A) (Yan et al. 2021). On the other hand, Zhu et al. (2020) identified five QTLs on chromosome 1AS, 2DL, 5AS, 5AL, and 7DS from Chinese elite lines. The number of mapped QTLs in the A, B, and D in the wheat sub-genomes were 192, 238, and 121, respectively. Eighty-one QTLs on chromosomes 3B, 58 QTLs on 5A, and 57 QTLs on chromosome 2D were the ones that showed the largest amount of the mapped regions (Venske et al. 2019). Among the reported QTLs, the most commonly mapped trait was type II resistance (41.5%) and the less mapped trait was type I (11.5%) resistance (Venske et al. 2019). No QTL was reported related to type V resistance. The type of resistance, sources

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of resistance, and corresponding chromosome locations are summarised in Table1 and Table S1 in the Electronic Supplementary Material (ESM).

Major FHB resistance genes/QTL identified by molecular markers. Nowadays, several FHB QTLs for various types of FHB resistance have been identified and located on the 21 wheat chromosomes (Buerstmayr et al. 2019; Jemanesh et al. 2019). *Fhb1* was the first QTL identified from the Chinese wheat Sumai 3 and confers to type II (pathogen spread) and type resistance (Anderson et al. 2007). Furthermore, *Fhb1* enhances the conversion of the DON toxin into a less toxic form called DON-3-glucoside playing a detoxifying role (Lemmens et al. 2005). This gene was sequenced and cloned by Rawat et al. (2016) and the *PFT* (pore-forming toxin-like) gene was responsible for the resistance at the *Fhb1* locus (Su et al. 2019). Recent studies showed the gene *TaHRC* was proposed as the candidate gene for *Fhb1* (Su et al. 2019; Li et al. 2019b). Most of (60.90%) the studies on resistant genes were focused on *Fhb1* because it is the most stable and has been found to limit the severity of the FHB disease tremendously. Besides, MAS is used to incorporate its resistance in host resistance breeding programmes (Buerstmayr et al. 2009). Xgwm533, Xbarc133, and Xgwm493 simple sequence repeat (SSR) markers have been used in mapping *Fhb1* (Cuthbert et al. 2006; Hao et al. 2020), however, the most important and most commonly used codominant marker for *Fhb1* is Xumn10 (Liu et al. 2019).

Fhb2 is an important QTL identified in a recombinant inbred mapping population from the cross of BW278 × AC Formost, and located on the chromosome arm 6BS. *Fhb2* confers type II resistance and studies carried out in the greenhouse showed that cultivars with *Fhb2* had a 56% less FHB severity index (Cuthbert et al. 2007). *Fhb2* was mapped between Xgwm133 and Xbarc79 with Xgwm644 SSR markers and located on the chromosome arm 6BS from Sumai-3 (Cuthbert et al. 2006) and it accounted for 21% of the variation (Yang et al. 2005). It was reported not only in Chinese landraces, but also in landrace sources from other countries including DH181 from Canada, Blackbird from Persia, Patton from the USA, Arina, and Apache from Europe (Bai et al. 2018).

Fhb3 was obtained from *Leymus racemosus,* a non-native wheat-related species. It is a useful QTL located on the chromosome arm 7A and was mapped between the gwm471 and gwm233 molecular mark-

Table 1. Major Fusarium head blight (FHB) resistance genes, resistant wheat germplasms and their flanking SSR molecular markers

Resistance gene/QTL	Resistant source	Marker name	Marker type	Chr.	Annealing temperature (°C)	Fragment (bp)[1]	Reference
Fhb1	Sumai-3	Xgwm493, Xgwm533	SSR	3BS	58	197	Xue et al. (2010)
	Wangshuibai	Xumn10	KASP	3BS	60	241–242	Liu et al. (2008)
Fhb2	Sumai-3, Wangshuibai, Ning894037	gwm133, gwm644	SSR	6BS	61	121	Cuthbert et al. (2007)
Fhb3	*Leymus racemosus*	gwm471, gwm233	SSR	7A	50	166	Xue et al. (2011)
Fhb4	Wangshuibai, Becker, and IL-95-1653	Xhbg226, Xgwm14	SSR	4BS	55	166	Cuthbert et al. (2006)
Fhb5	Wangshuibai, Wuhan1, Becker, IL 95-1653	Xgwm415, Xgwm304	SSR	5A	61	176	Xue et al. (2010)
Fhb6	*Elymus tukushiensis*	Xbarc133, Wg1s_snp1	SSR, KASP	1AS	51	125	Cainong et al. (2015)
Fhb7	*Thinopyrum*	Xcfa2240, XsdauK66	SSR	7DS	51	181	Guo et al. (2015)
Qfhb.nau-2B	CJ9306, Ning8940	Xwgrb156,1wgrb1410	SSR	3BS	60	125–190	Cuthbert et al. (2007)
Qfhs.ndsu-3AS	CM820	Xwgc501, Xwgc510	SSR	3A	60	100–125	Cuthbert et al. (2007)

[1]DNA fragment base pair

https://doi.org/10.17221/1/2022-CJGPB

ers. This QTL was identified in the wheat–*Leymus* cross line (Zhu et al. 2019b). Studies carried out in the greenhouse and field suggest that the cultivars with *Fhb3* had large levels of resistance, but mainly type II resistance. Nevertheless, the effects were not significantly different from Sumai-3. Some QTLs against FHB disease expansion were also found in the resistance evaluation to FHB in wild wheat relatives, including *Fhb3*, *Fhb6*, and *Fhb7*. *Fhb3* was derived from Daliancao 7r#1S (Qi et al. 2008). *Fhb4* (Qfhi. nau-4B), is an essential gene for type I resistance, and was mapped between the flanking markers, Xhbg226 and Xgwm149, and located on chromosome 4B (Xue et al. 2010), and was further located between Xmag8894 and Xmag8990 with a genetic distance of 0.14 cM (Jia et al. 2018). This QTL was fine-mapped with a recombinant inbred line population obtained from the cross of a susceptible parent (Nanda2419) and a resistant parent (Wangshuibai) (Jemanesh et al. 2019). Resistant cultivars carrying *Fhb4* showed 60% less infection than cultivars not carrying *Fhb4* (Xue et al. 2010). *Fhb4* is present in Wangshiubai (Xue et al. 2010), Wuhan1 and in several US germplasm sources, such as Becker and IL-95-1653 (Liu et al. 2009). *Fhb5* is identified in Wangshiubai and is located on the chromosome arm 4B (Xue et al. 2010). It is flanked by the Xgwm304 and Xgwm415 SSR molecular markers. This QTL is important for type-I resistance and accounts for 16.6–27% of the phenotypic variation for the FHB severity (Lin et al. 2006).

Fhb6 was mapped by QTL on the translocation line T1AL and located on chromosome 1AS. 1ASsLETs#1S was constructed by a cross between *Elymus tsukushiensis* and wheat (Cainong et al. 2016). Many genetic loci in wheat affect the FHB resistance though most only have minor effects, only a small number display a stable major effect on the FHB resistance (Guo et al. 2015). *Fhb7* is located on wheat chromosome 7DS which was derived from the alien species called *Thinopyrum ponticum*. *Fhb7* is a stable gene and it has a major QTL with a major effect on the FHB resistance (Zhang et al. 2011) which was located on 7LL (7DS) and 7EL2 (7D) by the e-chromosome substitution system of the *Thinopyrum elongatum*. Guo et al. (2015) located the gene within the range of markers Xsdauk66 and Xcfa240, with the genetic distance between the markers being 1.7 cM. Recently, *Fhb7* was mapped by Wang et al. (2020) to the distal end of the 7EL based on the recombinant inbred line population from a cross between an FHB-resistant line (7D/7E2) and an FHB susceptible line (7D/7E1). Table 1 presents the FHB-resistant genes identified in wheat cultivars using molecular markers and their flanking molecular markers.

Map-based cloning of FHB resistance genes. Among the seven *Fhb* resistance genes, *Fhb1* and *Fhb7* have been cloned so far. A pore-forming toxin-like gene at *Fhb1* was first cloned by map-based cloning and reported to confer FHB resistance in wheat (Rawart et al. 2016). *Fhb1* was discovered in the Chinese germplasm and is the most stable and has a major effect on the FHB resistance in wheat (Su et al. 2019). *Fhb1* was cloned recently by Li et al. (2019a) and Su et al. (2019) simultaneously. This creates an opportunity to obtain breeder-friendly molecular markers and improves the use of marker-assisted FHB resistance breeding (Yan et al. 2021). Resistance to FHB is controlled by many QTLs (polygenic), it can be affected by the environment and a direct MAS is difficult especially when we have QTLs with minor effects. Several studies have shown that the phenotypic selection was a more effective screening method for FHB as it integrates the unknown and known QTLs with their unknown interactions (Wilde et al. 2007, 2008; Miedaner et al. 2008). In an experiment that evaluated the Sumai-3 background, CM 82036 reported that the 5A and the QTL *Fhb-1*combination reduced the FHB symptoms by 55% and DON ones by 78% (Miedaner et al. 2006). Another study reported 17 QTLs with a good phenotyping approach, some of them influenced the FHB response, others the FDK and DON, some the FHB + FDK, and only four of them influenced all three traits (Szabó-Hevér et al. 2014).

Fhb7 was successfully cloned from the *Elymus* triticeae genome and its molecular resistance mechanisms were also characterised (Wang et al. 2020). In addition, *Fhb7* confers resistance to various *Fusarium* chemotypes such as *F. pseudograminearum* for crown rot and *F. asiaticum*, the most dominant FHB causing pathogen in southern China. Wang et al. (2020) confirmed that *Fhb7* transgenic plants found significantly smaller lesions than non-transgenic plants for all of the tested *Fusarium* species. This gene protects plants from *Fusarium* pathogens causing cytotoxic damage by enzymatic conversion and detoxifying trichothecene via de-epoxidation (Wang et al. 2020). Glutathione S-transferase 35 transcript T26102 was found recently (Guo et al. 2021), which was homologous to *Fhb7* and induced dramatically by *F. graminearum*. Homologues of *Fhb7* were also

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detected in several genera in *Triticeae*, including *Thinopyrum*, *Elymus*, *Leymus*, *Pseudoroegneria,* and *Roegneria*. To effectively use these resistance genes (*R* genes) and to better understand their mechanism of resistance in wheat, it is an important task to carry out map-based cloning and genome sequencing of these *R* genes.

Resistance mechanisms to FHB

Physical and physiological barriers to FHB. Plants, particularly cereal crops possess various resistance mechanisms that regulate FHB resistance including developmental, morphological, and physiological resistance traits. Morphological structures, such as the flower opening, plant height, (Gilsinger et al. 2005) ear compactness and heading date (Schmolke et al. 2005), and the presence of awns (Mesterházy 1995), have been associated with FHB resistance and influence the FHB development in wheat. Proline-rich proteins or phenols are chemicals or compounds in the physical barrier that may prevent the FHB infection. Resistant wheat cultivars may produce physical barriers to delay the mycelium growth within the wheat spike or release phenolic compounds and triticens that are poisonous to the disease causal organism. As a result, it can protect the wheat spike from quick desiccation during primary infection (Siranidou et al. 2013). Buerstmayr et al. (2021) found a higher induction of expression of the stress and diseased pathway genes in response to *F. graminearum* in the more susceptible lines. The inhibition of these genes in the resistant genotype after the pathogen infection might add to the remodelling of the cell wall during FHB pathogen attack, and, at the same time, the higher expression noticed in the susceptible infected spikes might contribute to the higher invasion of the pathogen in susceptible tissues (Biselli et al. 2018). This study showed the performance of Sumai 3 lines may depend on several defence mechanisms associated with cell wall biosynthesis and volatile organic compound emissions. Expressed products related to the detoxification of enzymes, synthesis of phytoalexins, production of antioxidants and antimicrobial substances, and cell wall modifications were recognised as inducible by *F. graminearum* infections, however, not all caused the differential induction in the susceptible and resistant cultivars, showing some of them participated in the FHB defence mechanisms (Biselli et al. 2018).

The biochemical events that occur during FHB attacks in resistant and susceptible near-isogenic lines are summarised in Figure 2. The basal FHB disease response involving miRNAome includes the alteration of the cell metabolism that causes the prohibition of photosynthesis, the targeting of the hormone signalling, including an auxin signal cascade, the activation of the cell wall strength, superoxide depression activities, and genes that show in pathogen resistance reactions are summarised by Biselli et al. (2018). Besides, they discovered several defence response genes that differentiated in the tested Near-isogenic lines (NILs), and they confirm the presence of crosstalk between the pathogen and the plant (Figure 1).

Role of pathogenesis-related genes, enzymes, and Phytohormones on FHB resistance. Defence response (*DR*) genes or pathogenesis-related (*PR*) genes have become potential sources of genetically manipulated resistance to FHB in wheat. FHB resistance in transgenic cultivars have shown that the expression of a pathogenesis related (PR) gene that is the same as thaumatin (the protein found in rice) (Fabre et al. 2020). Most of the *PR* genes found in both resistant and susceptible varieties which contain *Fhb1* from Sumai-3 and genes that encode for PR-4, PR-2, and PR-5 are displayed after wheat is colonised by *F. graminearum* (Fabre et al. 2020). The expression levels of PR-proteins and peroxidase can be measured by the northern blot analysis. Several *DR* genes exhibited to be induced in wheat spike during *F. graminearum* infections (Bernardo et al. 2007). Tang et al. (2020) explained that PR-4 and PR-5 proteins had much earlier and greater expression in resistant wheat cultivars (e.g., in Sumai-3) than in susceptible cultivars (e.g., in Wheaton) and could be detected 6 to 12 h after spike inoculation (Pritsch et al. 2016). Based on this, we can conclude that the induction of the FHB pathosystem defence gene expression correlates with *F. graminearum* resistance. Other genes that contribute to DON detoxification include *Ddna*, *EIN2* (Chen et al. 2009), *TaMetRS* (Zhu et al. 2016), *TaABCC3.1* (Walter et al. 2015), *HvUGT13248* (Li et al. 2015), *Bradi5g02780*; *Bradi5g03300* (Schweiger et al. 2013), *TaFROG* (Perochon et al. 2015) and *TaUGT12887* (Schweiger et al. 2013).

Several studies conducted extensive trials to evaluate the activities of the superoxide dismutase enzymes in susceptible and resistant genotypes (Tang et al. 2020) which were found significantly higher in spikes of the resistant genotypes than in the susceptible

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ones (Mauro et al. 2020). In the infected spikes of the resistant genotypes, the peroxidase activity increased until the 16th-day post-infection, but in susceptible genotypes, this enzyme started to decline post-infection. Zhang et al. (2012) analysed the protein expression of wheat under the condition of *Fhb1* resistance by using *Fhb1* and *Fhb1* free NILs. Nine proteins were found to be induced or upregulated in the resistance proximal gene line. The *Fhb7* enzyme neutralises DON by conjugating a glutathione S-transferase onto its toxic epoxide moiety. This explains the resistance conferred by *Fhb7* because DON is an important virulence factor required for Fusarium growth on infected tissues (Wang et al. 2020; Figure 2B). Figure 2 shows two paths of resistance mechanisms to FHB resistance in wheat.

Genetic studies have shown that ethylene (ET) signalling promotes FHB susceptibility while salicylic acid signalling promotes resistance in wheat (Chen et al. 2009). Lower expression of ethylene signalling and higher expression of the Jasmone acid gene have been reported to have an influence on wheat varieties having *Fhb1* (Xiao et al. 2013). Chen et al. (2009) found reducing the *EIN2* expression in wheat decreased the disease symptoms and DON accumulation in the grains. The transcriptome analysis suggested that the ethylene pathway was particularly induced in the FHB susceptible NAUH117 line, as compared to Wangshuibai, a resistant cultivar (Xiao et al. 2013). The abscisic acid signalling pathway has also been shown to favour *F. graminearum* invasion in wheat spikes (Wang et al. 2018).

Wheat breeding for resistance to FHB

Conventional breeding. Developing wheat resistance cultivars by using traditional crossbreeding methods with improved resistance is a key objective of wheat breeding programmes. In the last three decades, the search for host plant resistance based on phenotypic selection has made excellent progress in producing some wheat breeding lines with the best levels of FHB resistance. Up to now, Sumai-3 has been successfully used as a resistant parent in wheat-

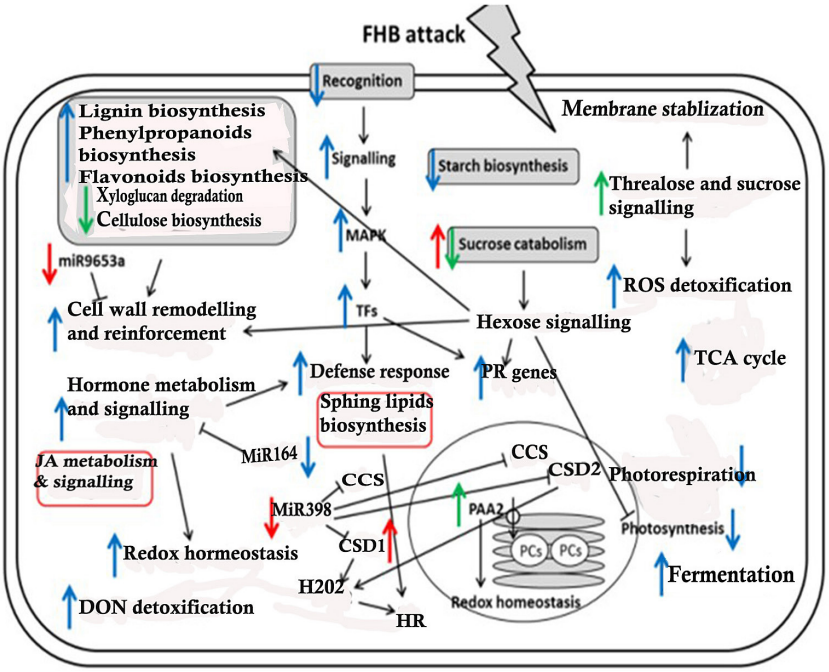

Figure 1. Summary of the biochemical events during an Fusarium head blight (FHB) attack in resistant and susceptible NILs
Blue arrows indicate modulation implicated in the basal defence response; red arrows indicate modulation after infection in the susceptible near-isogenic lines (NILs) only; green arrows indicate modulation after infection in the resistant 2DL+2-2618 NIL only and the involvement of 2DL specific resistance; red boxes indicate higher expression in S null 2-2890 with respect to R; red boxes indicate higher expression in S null 2-2890 with respect to R 2DL+ 2-2618 (Biselli et al. 2018); CCS – copper chaperone for superoxide dismutase; CSD – Cu/Zn superoxide dismutase; HR – hypersensitive response; MAPK – mitogen-activated protein kinase; PAA2 – P-Type ATPase 2; PCs – plastocyanins; TCA – tricarboxylic acid; TFs – transcription factors; PR – pathogenesis related; ROS – reactive oxygen synthesis (source: Biselli et al. 2018)

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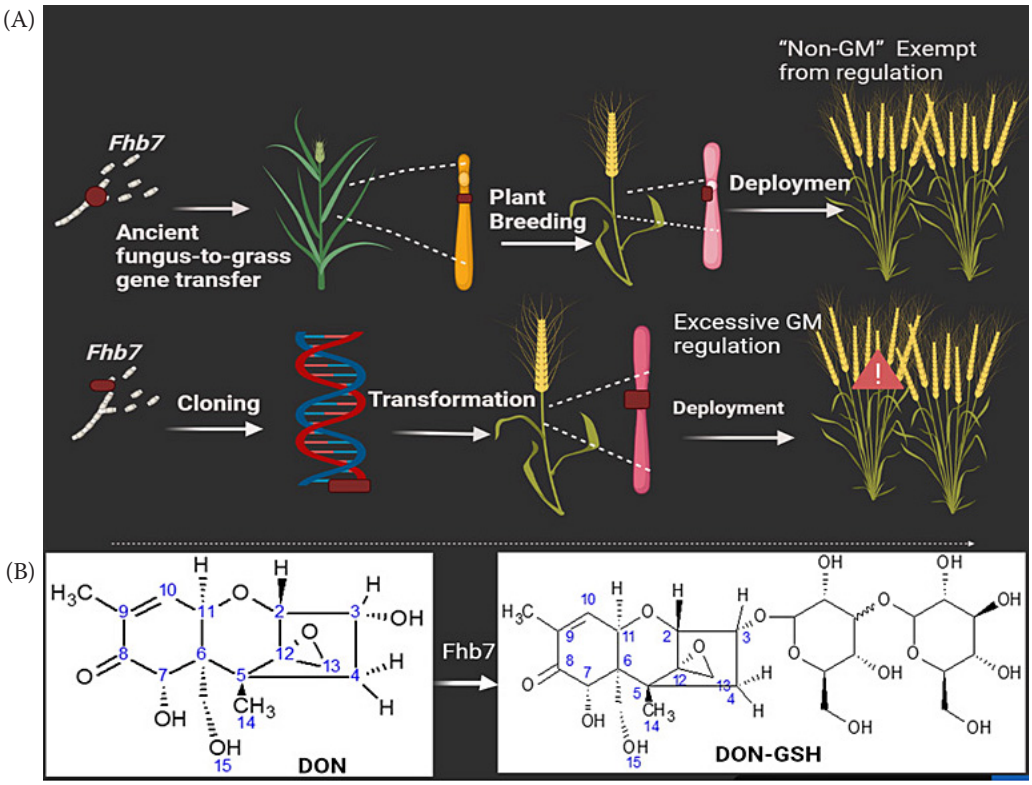

Figure 2. Two mechanisms of resistance to Fusarium head blight by a resistant wheat: the Fhb resistant *Fhb7* gene was introduced from Epichloe to wild wheat (*Thinopyrum*), and then from wild grass to common wheat to create the FHB resistant wheat cultivars (A), the Fhb7 enzyme detoxifies DON toxin by the formation of conjugating a glutathione (GSH) onto its toxic epoxide moiety (B); GM – genetically modified (source: Wang et al. 2020)

breeding programmes, and it is a major FHB resistant source for more than 20 modern wheat cultivars in the United States of America (US), Canada, Australia, and Japan (Hao et al. 2020). Chinese breeders successfully bred Sumai 3 from the hybrid offspring of Afu and Taiwan wheat which was considered a good source of FHB resistance worldwide. Since then, a lot of studies have been carried out on resistance breeding on Sumai 3 and Wangshuibai as donors, but no significant progress has been made, the main reason is that resistance to FHB and the comprehensive yield is difficult to coordinate. For example, Ning 7840, which was bred from Sumai No. 3 as a parent, had high resistance to FHB, but it was not directly used in production because of its high yield. Improved cultivars with moderate resistance to FHB and the comprehensive yield including Yangmai4, Yangmai5, Yangmai158, Ningmai 9, Tokai 63, Shinchunaga, Nobeokabouzu, and Nyu-Bai have been reported from China & Japan (Mesterházy 2017; Ma et al. 2019). Other ones such as Frontana and Encruzilhada are from Brazil, Ernie, Freedom, and Roane are from the United States (Liu et al. 2007; Jin et al. 2013), Chokwang is from Korea (Yang et al. 2005), and Arina, Renan, and Fundulea 201R are from Europe (Paillard et al. 2004; Somers et al. 2004). They have been extensively used as parents in breeding programmes (Liu et al. 2019; Mueller et al. 2019). Most of these lines have also been widely used as resistant parents in wheat breeding programmes in The International Maize and Wheat Improvement Center (CIMMYT), Europe, the US, and elsewhere. Where FHB threatens wheat production, breeding efforts have been conducted to identify sources of resistance from both exotic and native sources using conventional and molecular techniques (Yu et al. 2008; Duba et al. 2018). Most of the superior cultivars developed through conventional plant breeding strategies only showed moderate FHB resistance (Ma et al. 2020). Breeders should pay more attention to the genetic improvement of wheat resistance to FHB in the future. Table 2 presents FHB resistant germplasm and types of FHB resistance

Molecular marker-assisted breeding for FHB resistance. Marker-assisted breeding is a neutral and

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Table 2. Fusarium head blight (FHB) resistant wheat germplasms, molecular markers corresponding types of resistance and their chromosome locations in wheat

Resistance source	Marker	Resistance type	Chr.	References
Arina	Xgwm425-XmCCT.eAAG.2	type II	2A	Ma et al. (2006)
	Xwmc264-Xgwm155		3A	Paillard et al. (2004)
	Xgbx3480b-Xbcd907 g		4A	
	Xcdo545-Xgwm160		4A	
	Xgwm169-Xpsr966b		6A	
	Xgwm268-Xwmc44		1B	
	Xglk302b-Xgwm539		2D	
	bcd907c-Xgwm161		3D	
	Xcfd19b-Xgdm14b		6D	
	Xpsr915-Xcfd19a		6D	
	Xcfd19a-Xcfd47		6D	
Alondra	Xgwm146-Xgwm611	type II	7B	Jia et al. (2005)
	Xgwm296-Xgwm261		2D	Shen et al. (2003)
	Xgwm190-Xgwm358		5D	Jia et al. (2005)
Annong 8455	mGCG.pGTG223-Xwmc617.2	type II	4A	Ma et al. (2006)
	XFHBSTS1A-160		1A	Guo et al. (2015)
AC Foremost	Xwmc165	type I	3A	Yang et al. (2005)
CASS94	Xgwm539	type II	2D	Somers et al. (2003)
Chinese Spring	XmCGTA.pACT236-XmACAG.pACT134	type II	2D	Ma et al. (2006)
	XmCGAC.pTGC102-XmTGC.pTGC70		2D	
	Xcfd84-Xwmc331		4D	
CJ9306	gwm493-Xgwm533-2	type II	3A	Jiang et al. (2006)
	Xgwm157-Xgwm539		2D	
CM-82036	XgluB1	type II	1B	Buerstmayr et al. (2002)
	Xgwm293-Xgwm304		4D	Buerstmayr et al. (2003)
Cansas	XE38M52-378-Xgwm131	type II	1B	Klahr et al. (2006)
	XE35M52-331-XS25M 20-245		5B	
	XE33M57-457-Xgwm645		3D	
Chokwang	Xgwm533- Xgwm493	type II, III	3A	Yang et al. (2005)
	Xbarc1096	type II	4B	
	Xbarc 239	type II	5D	
DH181	Xgwm533	type I, II	3A	Yang et al. (2005)
	Xwmc612	type I	3A	
	Xwmc397	type I, II	6B	
	Xwmc526	type II	7B	
	Xgwm539	type I	2D	
	Xwmc144	type II	2D	
	Xwmc526		7D	
	Xwmc526		7D	
Dream	XP77M51_430-XS66M55_242	type II	6A	Schmolke et al. (2005)
	XP74M53_272-S25M12_206		2B	
	XS25M15_187-XS23M21_497		7B	
Emus	Xs13m26_4	type II	2A	Steiner et al. (2004)

https://doi.org/10.17221/1/2022-CJGPB

Table 2 to be continued

Resistance source	Marker	Resistance type	Chr.	References
Ernie	E8M4_6	type III	3A	Liu et al. (2005)
	Xgwm276b		2B	
Forno	gwm371-Xpsr120a	type II	5B	Paillard et al. (2004)
	Xpsr1201-Xgwm371		5B	
Frontana	Xs13m25_8–Xs24m15_6	type I	2B	Steiner et al. (2004)
	gwm720-Xgwm112		3A	Mardi et al. (2005)
	Xdupw227-Xgwm720		3A	Steiner et al. (2004)
	Xs13m25_9		4B	
	Xs23m14_4		6B	
	Xs23m14_4		6B	
	Xe77m47_22–Xgwm233	type I	7A	Mardi et al. (2005)
Fundulea201R	Xgwm674-Xbarc6	type II	3A	Shen et al. (2003)
	Xbarc8-Xgwm131		1B	
Goldfield	Xbarc200-Xgwm210	type I	2B	Gilsinger et al. (2005)
Lynx	XP78M51_237-XS26M23_365	type II	2A	Schmolke et al. (2005)
Massey	Xbarc334, Xgwm192	type II	7A	Liu et al. (2013)
Maringa	Xgwm261	type III	2D	Somers et al. (2003)
	Xgwm533-Xgwm493		3A	
	Xgwm566	type I	3A	
Nanda2419	Xgwm533-2-Xwmc054-1	type I	3A	Lin et al. (2006)
	Xwmc501-2-Xwmc161		4A	
	Xwmc338-2–Xwmc83	type II	7A	
	Xs1021 m		2B	
	Xgwm469		6B	
	Xbarc126-2-Xwmc476	type I	7B	
	Xgwm437-Xwmc488		7D	
NC-Neuse	Qfhb.nc-1A	type II	1A	Petersen et al. (2016)
	KASP markers Qfhb.nc-1A		1B	
ND2603	Xbcd941	type II	3A	Anderson et al. (2001)
Ning 7840	XmGTG.pAG225-Xbarc28	type II	1A	Ma et al. (2006)
	Xgwm120		2B	Zhou et al. (2003)
	Xgwm614		2A	
	gwm533-Xcfd79		2D	Kang et al. (2011)
Ning894037	Xgwm88-Xgwm644	type II	6B	Shen et al. (2003)
Patterson	Xbarc59	type II	5B	Paillard et al. (2004)
	Xgwm341-Xgdm8		3D	Shen et al. (2003)
Renan	Xgwm311-Xgwm382	type II	2A	Gervais et al. (2003)
	Xgwm374		2B	
	Xcfd29		5D	
	Xcfd42		6D	
Remus	Xs12 m 25_14-Xs24m17_2	type I	3A	Steiner et al. (2004)
Ritmo	XS16M22-162-Xwhs2001	type I	1D	Klahr et al. (2006)
	XE35M59-107-XE38M52-441	type II	3A	
	XS23M21-271-XS18M22-369		7A	

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Table 2 to be continued

Resistance source	Marker	Resistance type	Chr.	References
Seri82	Xe38m50_10-Xe32m65_10	type I	1B	Mardi et al. (2005)
Stoa	XksuH16	type II	2A	Anderson et al. (2001)
Sumai 3	XeagcMcta.1	type II	3A	Anderson et al. (2001)
	Xcdo981		3A	Waldron et al. (1999)
	XksuH4		6A	Anderson et al. (2001)
	Xbcd331		6B	Waldron et al. (1999)
	Xcdo524		6B	
	Xbarc101-Xbcd1383		6B	Anderson et al. (2001)
Wangshuibai	Xcfa2086-Xgwm311	type II	2A	Paillard et al. (2004)
	Xbarc312-Xbarc302		2A	Zhou et al. (2003)
	Xwmc181-Xaf12	type I	2D	Lin et al. (2006)
	Xgwm533-3	type II	3A	
	Xwmc349-Xgwm149	type I	4B	Lin et al. (2006)
	Xgwm513-Xbarc20		4B	
	XmCCA.eAAG.2-Xgwm156	type II	4D	Ma et al. (2006)
	Xgwm304-Xbarc56	type I	4D	Lin et al. (2004)
	Xwmc539		6B	
	Xgwm133-Xgwm191	type II	6B	Jia et al. (2005)
	Xgwm276-Xgwm282		7A	
	Xwms1083		7A	Zhou et al. (2003)
Wuhan-1	Xgwm539	type II	2D	Somers et al. (2003)
W14	Xbarc117-Xbarc186	type I	4D	Chen et al. (2006)

DNA-based approach that is not affected by environmental factors. It has become a promising option to FHB resistance development because of the following reasons: reduces cost, speeds up the breeding process, increases breeding accuracy, and reduces the workload of resistance evaluation for plant breeders (Buerstmayr et al. 2009). The application of the *Fhb1* gene in the molecular marker-assisted breeding of resistance to FHB at home and abroad was mainly focused on. Xie et al. (2007) introduced the *Fhb1* gene into Australian varieties and reported that the resistance of the progenies was significantly higher than that of the recipient parents. Fox et al. (2013) developed a moderately resistant cultivar Cardale by using linkage marker-assisted selection of resistance sites on *Fhb1* and 5A. Since 1999, more than 20 spring wheat varieties have been bred and planted widely in the United States and Canada using improved Sumai 3 as parents. It has been reported that CIMMYT researchers have broken the adverse linkage between *Fhb1* and *Sr2* using a large population of binding molecular markers, and recombinant materials (Zhang et al. 2016; Buerstmayr et al. 2019) carrying both resistance genes have been created. Zhang et al.(2016) used Ningmai 9, Shengxuan 6, Jianyang 798, Jianyang 84, Sumai 3, and Ningmai13 as *Fhb1* gene donors, and backcross progenies carrying *Fhb1* were obtained by crossing and backcrossing with Zhoumai16 dwarf male-sterile wheat near-isogenic lines with high susceptibility to FHB and combining with a marker-assisted selection. Based on MAS using the SSR markers *Xbarc147* and *Xgwm389*, Zhou et al. (2020) reported a reduction in the percentage of scabbed spikelets from 70–80% to 30–40% for improved wheat lines carrying *Fhb1* compared to plants without *Fhb1* after introducing *Fhb1* from Ning 7840.

Role of chromosome engineering for resistance to FHB. Chromosome engineering (CE) and transgenic breeding are effective ways of creating new resistance sources. Artificial chromosome engineering in plants was summarised by Houben and Schubert (2007). *Fhb6*, a major resistance gene, was successfully mapped using chromosome engineering

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by from the alien species *E. tsukushiensis* into FHB resistance wheat by Cainong et al. (2015).

Fhb6 was mapped at the chromosome arm 1E ts #1S of *E. tsukushiensis*. A good deal of wheat genetic resource evaluation studies have been conducted in China and Japan, and reported *E. tsukushiensis* as a potential source of type I and type II resistance to FHB (Weng & Liu 1989). Guo et al. (2015) characterised and developed secondary 7DS.7el 2 L artificially cultivated lines with shortened *Th. ponticum* segments carrying *Fhb7*. A distant hybridisation binding molecular marker has been used to transfer chromosome segments carrying the *Fhb7* gene into wheat, which enhances the resistance to FHB which does not have a negative effect on the yield. More recently, derivative glutathione S-transferase transcript T26102, which was homologous to *Fhb7* and induced dramatically by *F. graminearum* was successfully applied by the chromatin derived from diploid *Th. elongatum*, which confers wheat with high-level FHB resistance independent of *Fhb7* (Guo et al. 2021). Many various transgenic approaches have been explored to produce crops that show enhanced FHB resistance. Nowadays, genetic engineering and biotechnology provide a useful approach for manipulating crop disease resistance and susceptibility and are becoming attractive alternatives to durable the FHB resistance.

Mutation breeding for FHB resistance. A mutation is an effective method in creating new resistance materials. Compared to methods of crossbreeding, mutagenesis enables modification of one or a few characteristics in an otherwise promising cultivar without significantly altering the remaining genetic background. However, breeding for FHB resistance is very complex: disease resistance is a quantitative trait, and influenced by biotic and abiotic stress (Arabi et al. 2019; Eid 2019). A high degree of new genetic change is frequently undertaken in crops using induced mutagenesis, mainly by ethyl methanesulfonate, sodium azide chemicals, or through gamma radiation (Cheng et al. 2015; Hussain et al. 2018). Since host resistance to FHB is controlled by polygenics and there is no complete resistant wheat cultivar against FHB, the use of alternative approaches to search for new genetic resistance is needed. Such approaches include the use of forwarding genetic screening to identify resistant mutants to FHB. In a recent genetic screening study conducted to identify resistant wheat mutants to FHB from M_4 generation induced by EMS, 74 mutant lines had resistance against FHB spread (Chhabra et al. 2021) and 30 of these lines were found to have a low DON content when inoculated with *F. graminearum*. In addition, a two year extensive screening programme under greenhouse conditions found that 10 M_6 mutant lines had a significantly smaller FHB spread, and seven lines showed lower DON contents (Chhabra et al. 2021).

Application of genomic selection (GS) in breeding for FHB resistance. GS has been employed in wheat breeding for resistance in different countries including Canada and the US. Rutkoski et al. (2012) evaluated the accuracy of GS models for FHB-related parameters using 170 wheat lines obtained from different countries and more than 2 000 SSR and diversity array technology markers and concluded that genomic selection is a promising breeding method for resistance improvement in wheat. Verges et al. (2020) recently reported the beneficial role of GS in the accurate precision of FHB parameters in a training population. SNP molecular markers flanking *Fhb1* locus were developed from Ning 7840/Clark (Bernardo et al. 2007). The gene-specific Kompetitive allele specific PCR (KASP) marker, named PFT_KASP, has been developed and validated to diagnose *Fhb1* (Singh et al. 2019). KASP markers linked to *Fhb1* have been developed (Steiner et al. 2017; Su et al. 2018). A recent study reported positive results with Genome-Wide Association Studies and Genomic Selection (GWAS-GS) for DON and disease scabbed kernel under a forward GS scheme, where a set of regional lines with known DON values becomes the TP (training population) to calculate the genomic estimated breeding values for wheat breeding lines that do not have any FHB phenotyping evaluation yet (Verges et al. 2021). This study confirms that a reduced marker number decreases the genotyping costs and will accelerate the breeding process.

Discussion and future perspectives

FHB is a severe and chronic disease of cereal grains particularly wheat. FHB causes extensive yield reduction and deteriorates the grain quality through its contamination with a plethora of mycotoxins. The toxins produced by Fusarium fungi include DON, T2 toxin, HT-2 toxin, serpentine toxins, nivalenol, and zearalenone (van der Fels-Klerx & Stratakou 2010). Many fungicides are effective in preventing the FHB pathogen and reduce mycotoxin contamination in wheat, but there are concerns related to the pathogen resistance and environmental issues. Employing genetically resistant cultivars remains the safest,

https://doi.org/10.17221/1/2022-CJGPB

effective, and most economical approach to reducing the losses caused by the pathogen. However, the contradiction between FHB resistance and a high yield is even now a bottleneck problem for wheat breeding globally. Besides, host resistance is of a polygenic trait. For this reason, it is very urgent to strengthen the genetic and breeding research of host resistance to FHB and cultivate wheat cultivars with a synergetic improvement of the yield and resistance. To obtain improved cultivars resistant to FHB, many resistant identification methods have been used and numerous breeding resources have been screened using molecular markers in the last decades. More than 400 resistant QTLs have been identified so far (Buerstmayr et al. 2009; Jia et al. 2018; Ma et al. 2020), and mainly located on chromosomes 5A, 3B, 6B, 6D, and 7D (Lin et al. 2004; Ren et al. 2019).

The aims of mapping FHB resistance genes/QTLs with polymerase chain reaction (PCR)-based molecular markers is to improve the efficiency of selecting FHB-resistant varieties (Guo et al. 2015). Extensive studies have been undertaken on the pathogenic mechanism of the wheat FHB pathogen, identification and screening of resistant resources, cloning of resistant genes, and breeding of resistant varieties, and important progress has been made particularly in Sumai-3 and other closely related lines. There is no breakthrough in resistance breeding by using common wheat as a donor. Due to this, many breeders at home and abroad are searching to further explore noble resistance sources from wheat relatives and creating additional lines. Though the effect is not huge, a number of resistance genes or QTLs have been successfully transferred from exotic species to wheat relatives, including *Fhb3* to *Leymus macrophylla*, *Fhb6* to *Elymus dahuricus* and *Fhb7* to *Thinopyrum elongatum.* So, many sources of resistance to FHB have been identified from different wheat-growing countries to have moderate to high levels of FHB resistance (Chen et al. 1995, 2005; Weng et al. 1995; Guo et al. 2015; Brisco et al. 2017), but there is no completely resistant variety so far. Therefore, the research and utilisation of resistance genes in related species should be further strengthened to find genes with a greater effect and enrich the diversity of resistant genes so that it will help to set a foundation for a breakthrough in host resistance breeding.

As visual symptoms are less reliable, breeding works should concentrate on the FDK and DON as this method enhances the efficiency of selection, and less DON contamination, further enhances food safety (Mesterházy et al. 2015), and DON contamination is mainly genotype-specific, should be measured as type III resistance. The most important parameters and requirements that should be considered were summarised by (Mesterházy 1997). The local resistance sources are invaluable as they are easily adaptable and previous studies identified many moderately and high resistant cultivars, genotypes, and landraces with the best resistance (Snijders 1990; Brown-Guedira et al. 2008), and recommended that, in a variety resistance evaluation using artificial inoculation and in the variety registration process, they should be included to realise the food safety issues. Besides, an FHB resistance evaluation and breeding for resistance are difficult processes. The good thing for screening resistance is that we do have horizontal and non-race-specific resistance, and we have to analyse all the parameters.

Furthermore, wheat FHB susceptibility genes, pathogen, host symbiosis, and in addition to the employment of gene editing technologies in wheat resistance breeding, genome assisted selection methods, such as GS and MAS with phenotype selection together with rapid disease resistance gene discovery, and cloning technologies, like the clustered regularly interspaced short palindromic repeats-associated protein 9 (CRISPR-Cas9) (Li et al. 2021), will be a suitable strategy to advance resistance breeding for FHB which all need to be further studied.

In addition, coordinated efforts are needed among regional and global institutions including the US Barley and Wheat Scab Initiative (USWBSI) and the Chinese FHBRPMWS (FHB Remote Monitoring and Warning System) (http://www.cebao wang.com/wheat monitor/), both are actively developing early warning short messages or email warnings to subscribers when environmental conditions are suitable for FHB development. Such alert notifications provide accurate forecasting systems that can help extension experts and farmers in decision-making and thereby minimising the risk of disease. Finally, this will help to control the incidence and spread of FHB, at the same time permitting growers to stay alert.

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